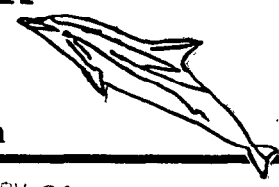
Department of the Planet Earth

701 E Street, SE - Suite 200, Washington, DC 20003

(301) 475-8366 - planetearth@erols.com; www.deptplanetearth.com



Ms. Judy Kidwell
Division of Dockets Management
Office of Management Programs
Office of Management
Food and Drug Administration
5630 Fishers Lane, Room 1061
Rockville, Md. 20857

November 14, 2005

Re: Docket No.: 2005P-0377/CP 1 (An additional study.)

Dear Ms. Kidwell,

Enclosed is a high quality measurement study of levels of aluminum, magnesium, and phosphorous in the brain of AD patients compared that of controls by Andrasi. Clearly, aluminum is elevated in the AD brain.

1. As you can see, there is a rather dramatic increase of aluminum in the five brain regions studied, accompanied by a depression of both magnesium and phosphorous.

2. Aluminum is competitive with magnesium, but also with phosphorous. (Indeed, DeLoncle found that aluminum could be chelated from the brain of laboratory rats with a combination of magnesium D-aspartate combined with sodium L-glutamate). (1)

3. The authors suggested that the depression of phosphorous might largely be caused by depletion of myelin (which is also generated by aluminum exposure) (2)(4), though aluminum is also competitive with phosphorous in absorption.

4. One note: While increased brain aluminum has been been shown to directly cause brain cell death, the metal also may cause brain cell death indirectly: i.e. through damage to bone marrow cells producing red blood cells (3) or perhaps damage to the liver (4).

❑ <u>Authors Out of Date in Their Supporting Literature Search for Causation</u>

While this seems a brilliant brain measurement study, the authors are citing epidemiology and other supporting literature from 15 years ago, and are

not up-to-date. Being from Hungary with less access to libraries, this is not unexpected. For example, they cite Michel's epidemiology study of 1990. But, that was withdrawn by the authors in 1992 after they discovered that the governmental measurement of drinking water aluminum were dramatically in error. Fleming's study on ferritin is also in dispute. And very many high quality epidemiology studies are available since 1990.

The authors are still arguing about the neurofibrillary deposits, which are not key to dementia as compared to brain atrophy. And they have missed all the relevant recent laboratory animal studies which give insight on how aluminum kills brain cells. Obviously, measurement is their speciality rather than epidemiology or in vivo studies.

Based on their quite incomplete and dated (i.e. 15 years ago) literature search, the authors conclude that a "role for Al in AD is an unproven and controversial hypothesis". This might have been a possible conclusion from literature of 1990, but certainly not today. Indeed in 1995, DRC McLachlan reached entirely different conclusions from the evidence.

But, it is clear from this well controlled study by competent measurement experts that aluminum is elevated in at least the five areas studied of the brain of the 77 year old AD patients by 2 to 7- fold compared to the 61 year old controls, while magnesium and phosphorous are reduced.

With best regards,

Erik Jansson

Erik Jansson, Exec. Dir.

(1) R. Deloncle et al, Aluminum L-glutamate complex in rat brain cortex: in vivo prevention of aluminum deposit by magnesium D-aspartate, Brain Res 946 (2002) 247-52
(2) S.V. Verstraeten et al, Myelin is a preferential target of aluminum-mediated oxidative damage, Arch Biochem Biophys 344 (1997) 289-94
(3) A.L. Florence et al, An experimental animal model of aluminum overload, Neurodegeneration 3 (1994) 315-23
(4) A.C. Miu et al, A behavioral and ultrastructural dissection of the interference of aluminum with aging, J Alzheimer's Dis 6 (2004) 315-28